PHYSIOLOGICAL VARIATIONS AND TENDENCY OF ACUTE MOUNTAIN SICKNESS IN TREKKERS

SYNOPSIS

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BY

UMASANKAR MOHANTY

GUIDE
Dr. H. NAGALINGAPPA
CHAIRMAN AND DIRECTOR
DEPARTMENT OF P.G. STUDIES AND RESEARCH IN PHYSICAL EDUCATION AND SPORTS
MANGALORE UNIVERSITY
INDIA

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CHAPTER I

INTRODUCTION

Few sights are more breathtaking and Inspirational than a mountain peak. The number of people traveling for mountain trekking has risen enormously. Without special climbing ability these climbers are exposed to the altitudes they are not used to in their usual lifestyle (Peacock 1998). Both the high altitude physiologist and the intensivist are challenged by the human organism in a hypoxic environment and the true research potentials presented by high altitude, where the body is subjected to an essentially isolated hypoxic challenge, are only just beginning to be realized (Mason 2000). Mountain climbing can also be a problem for people with cardiopulmonary disease. For most people altitude sickness is little more than an inconvenience but every year some die and often the victims are fit, enthusiastic, audacious and physically hardworking. For most the condition is trivial and consists of headache, nausea, anorexia, insomnia, giddiness, and ataxia, and lassitude, shortness of breath at rest, irritability, and personality change. More severe cases show peripheral edema or the rapidly fatal forms of pulmonary or cerebral edema. The nature of the mountain environment can present a number of challenges for mountaineers. Such challenges range from rock fall, high altitude pulmonary edema, high altitude cerebral edema, and sudden cardiac death (Windsor et al 2009, Schoffl et al 2009).

1.1. Mountain Environment

Although the percentage of oxygen in inspired air is constant at different altitudes, the fall in atmospheric pressure of higher altitude decreases the partial pressure of inspired oxygen hence the driving pressure for gas exchange in the lungs. The stress of the altitude is primarily due to the decrease in (PO_2) partial pressure of oxygen and not barometric pressure (Peacock 1998, Honigman et al 1993, Wagner 1996, Wagner 2000).

PO_2= 0.2094×barometric pressure (Pb-6.3)

PO_2= Inspired O_2 tension

0.2094= Atmospheric concentration

6.3= Water vapor pressure

The following are the presentations of the barometric pressure.

Sea Level= 760 mmHg

At 3048m= 510 mmHg

At 5486m= 380mmHg

Mt. Everest= 250mmHg

As Oxygen is 21% dry air, the inspired Oxygen pressure is 0.21×(100-6.3)= 19.6 kPa at sea level. The atmospheric pressure is the sum total of the partial pressures of the constituent gases, Oxygen and Nitrogen and also the partial pressure of water vapour (6.3 Pa at 370^0 C). The reduction of partial pressure leads to arterial hypoxia. Exposure to even mild hypoxia stimulates the peripheral hemoreceptors due to increase in activity in the carotid sinus (Honigman , Theis , Koziol-McLain et al 1993).

1.2. Acute Mountain Sickness

Behind the innocence and majesty of mountain climbing lies a sleeping evil, acute mountain sickness (AMS). AMS is a frequent cause of morbidity and occasionally mortality and affects 25% of travelers journing to altitudes (Honigman, Theis, Koziol-McLain et al 1993). The Acute Mountain Sickness comprises of frontal headaches, lassitude, Insomnia, and peripheral edema. As far as the cardiovascular system is concerned there is an increase in the submaximal HR, Increase in the submaximal cardiac output, stroke volume is same or decreased. In the pulmonary system there is hyperventilation and the body fluids become alkaline due to CO_2 elimination.

Although the precise mechanisms of AMS and its related sequelae remain elusive, a low hypoxic ventilatory drive, water retention, and increased vascular permeability are recognized as the most important pathophysiologic Factors(Johnson and Rock 1988). Arterial oxygenation decreases as a function of the hypobaria at terrestrial altitude, and thus tissue hypoxia is considered a trigger for these changes (Johnson and Rock 1988).The source of membrane damage, which may be more pronounced in subjects presenting with AMS, may be linked to an increased activity of oxygen-free radicals. Physical symptoms associated with upper respiratory and gastrointestinal tract illnesses are ubiquitous among mountaineers who ascend to high altitude (Beall 2001, Cremona, Asnaghi, Baderna et al 2002, di Prampero and Ferretti 1990).

Statement of the problem

The mountain trekkers are prone for physiological variations. The purpose of the study was to find out the physiological variations for the trekkers and the tendency of Acute Mountain Sickness for the trekkers.

1.3. Hypothesis

Based on available literature and the understanding gained after analyzing the past results the following hypotheses were formulated:

1.3.1. There will be significant increase in Systolic Blood Pressure after trekking.

1.3.2. There will be significant increase in Diastolic Blood Pressure after trekking .

1.3.3. There will be significant increase in Pulse Rate after trekking.

1.3.4. There will be significant decrease in Oxygen Saturation after trekking

1.3.5. There will be significant change in Acute Mountain Sickness scores after trekking .

1.4. Null hypothesis

1.4.1. There will be no significant increase in Systolic Blood Pressure after trekking.

1.4.2. There will be no significant increase in Diastolic Blood Pressure after trekking.

1.4.3. There will be no significant increase in Pulse Rate after trekking.

1.4.4. There will be no significant decrease in Oxygen Saturation after trekking.

1.4.5. There will be no significant change in Acute Mountain Sickness scores after trekking.

Definitions and Explanations of Terms

1.5. Trekking

It is derived from the South African Dutch word trek (noun), trekken (verb), (literally meaning to "pull, travel"), became a word in English language in mid 19th century describing a long arduous journey undertaken on foot in areas where common means of transport is generally not available(Cox, 2003).

1.6. Mountaineering or mountain climbing

It is the sport, hobby or profession of hiking, skiing, and climbing mountains. While mountaineering began as attempts to reach the highest point of unclimbed mountains it has branched into specializations that address different aspects of the mountain and consists of three areas: rock-craft, snow-craft and skiing, depending on whether the route chosen is over rock, snow or ice (Cox, 2003).

1.7. Pulse Oximeter

It is a medical device that indirectly measures the amount of oxygen in a patient's blood (as opposed to measuring oxygen saturation directly through a blood sample) and changes in blood volume in the skin, producing a photoplethysmograph. It is often attached to a medical monitor so staff can see a patient's oxygenation at all times. Most monitors also display the heart rate(di Prampero and Ferretti 1990).

1.8. Heart Rate

Heart Rate is a term used to describe the frequency of the cardiac cycle. It is considered one of the four vital signs. Usually it is calculated as the number of contractions (heart beats) of the heart in one minute and expressed as "beats per minute" (bpm). The heart beats up to 120 times per minute in childhood. When resting, the adult human heart beats at about 70 bpm (males) and 75 bpm (females), but this rate varies among people. However, the resting heart rate can be significantly lower in athletes. The infant/neonatal rate of heartbeat is around 130-150 bpm, the toddler's about 100–130 bpm, the older child's about 90–110 bpm, and the adolescent's about 80–100 bpm (Shephard, Bouhlel, Vandewalle & Monod 1988).

1.9. Blood pressure

Blood pressure refers to the force exerted by circulating blood on the walls of blood vessels, and constitutes one of the principal vital signs. The pressure of the circulating blood decreases as blood moves through arteries, arterioles, capillaries, and veins; the term *blood pressure* generally refers to arterial blood pressure, i.e., the pressure in the larger arteries (Shephard, Bouhlel , Vandewalle & Monod 1988).

Blood pressure is most commonly measured via a sphygmomanometer, which uses the height of a column of mercury to reflect the circulating pressure. Although many modern blood pressure devices no longer use mercury, blood pressure values are still universally reported in millimetres of mercury (mmHg).The systolic pressure is defined as the peak pressure in the arteries, which occurs near the beginning of the cardiac cycle; the diastolic pressure is the lowest pressure (at the resting phase of the cardiac cycle). The average pressure throughout the cardiac cycle is reported as

mean arterial pressure; the pulse pressure reflects the difference between the maximum and minimum pressures measured (Shephard, Bouhlel, Vandewalle & Monod 1988).

1.10. Oxygen saturation

Oxygen saturation (SO_2) measures the percentage of hemoglobin binding sites in the bloodstream occupied by oxygen. At low partial pressures of oxygen, most hemoglobin is deoxygenated. At around 90% (the value varies according to the clinical context) oxygen saturation increases according to an Oxygen-hemoglobin dissociation curve and approaches 100% at partial oxygen pressures of greater than 10 k Pa. A pulse oximeter relies on the light absorption characteristics of saturated hemoglobin to give an indication of oxygen saturation (Wagner 1996).

1.11. Acute Mountain Sickness

Acute mountain sickness consists of a constellation of symptoms in the context of a recent gain in altitude. These include headache; anorexia, nausea or vomiting; fatigue or weakness; dizziness or and difficulty sleeping (Wagner 1996).

1.12. Significance of the study

1.12.1. The result of this study helps to understand the variation in the heart rate, breath rate, blood pressure and O_2 saturation in trekkers.

1.12.2. The study helps to compare the Acute Mountain Sickness to the changing physiological parameters.

1.12.3. The results of the study helps to train the mountain trekkers to train themselves in order to adjust with the changing physiology.

1.13. Delimitations

1.13.1. The study was delimited to 400 trekkers.

1.13.2. The study was delimited to the same group of subjects for pre and post trekkers.

1.14. Limitations

1.14.1. The Acute Mountain Sickness was measured with the Questionnaire and the responses obtained were more subjective. Being subjective is one of the limitations of the study.

1.14.2. Non cooperation by the trekkers was one of the limitations as some trekkers felt that the physiological measurements are wastage of their time.

1.15 USEFULNESS OF THE STUDY

1.15.1. The result of this study helped to understand the variation in the heart rate, breath rate, blood pressure and O_2 saturation in trekkers.

1.15.2. The study helped to compare the Acute Mountain Sickness to the changing physiological parameters.

1.15.3. The results of the study helped to train the mountain trekkers themselves in order to adjust with the changing physiology.

1.15.4. This research facilitated the percentage of hazards for the trekkers and the physiological trainings they require to adopt with the challenging environment.

CHAPTER II

LITERATURE REVIEW

In this chapter an extensive literature survey was carried out pertinent to the current research work. Reviews from 1985 to 2010 are covered and the review is supportive to the need of this research work.

Heggie and Jorgenson (2010) stated that, the nature of the mountain environment can present a number of challenges for mountaineers. Such challenges range from rockfall, high altitude pulmonary edema, high altitude cerebral edema and sudden cardiac death. The high-altitude dramatically influences human response during exercise and dramatically reduces its endurance performance. Hypoxia is the main single factor which influenced very complex adaptations called acclimatization. Enhanced neural stimulation from baroreceptors and sympatoadrenal activity influences complex changes of gas exchange (Wagner 2001), blood oxygenation and acid-base balance through reducing partial pressure of CO_2 (PCO_2) and additional extraction of bicarbonate (HCO_3) by kidneys (Smith, Dempsey and Hornbein 2001) .

At sea level carbon dioxide is the main stimulus to ventilation. At altitude hypoxia does increase ventilation, but usually only when the inspired oxygen pressure is reduced to about 13.3 kPa (3000 m altitude). At this inspired oxygen pressure the alveolar oxygen pressure is about 8 kPa, and with further increases in hypoxia ventilation rises exponentially. This hypoxic ventilatory response is mediated by the carotid body, and response varies widely among subjects. Interestingly, however, the ability to tolerate

altitude does not seem to relate to the presence of a brisk hypoxic ventilatory response (Peacock 1998, di Prampero & Ferretti 1990).

In the systemic circulation hypoxia acts as a vasodilator, but in the pulmonary circulation it is a vasoconstrictor. The purpose of hypoxic pulmonary vasoconstriction is unclear. It may help match ventilation and perfusion within the lung, but in hypoxia of altitude the reflex leads to pulmonary hypertension and is associated with high altitude pulmonary edema (Peacock 1998, Shephard , Bouhlel , Vandewalle & Monod 1988).

At sea level gaseous diffusion is probably limited by ventilation-perfusion matching in the lung. At high altitude, however, the alveolar-arterial difference for oxygen is higher than would be predicted from the measured ventilation-perfusion inequality. This is because the decreased driving pressure for oxygen from alveolar gas into arterial blood is insufficient to fully oxygenate the blood as it passes through the pulmonary capillaries. This is more evident on exercise as cardiac output increases and blood spends less time at the gas exchanging surface (diffusion limitation) (Wagner PD.1996).

The heart works remarkably well at altitude. Initially there is an increase in cardiac output in relation to physical work but later this settles to sea level values. At all times there is increased heart rate and decreased stroke volume for a given level of work, though the maximum obtainable heart rate falls as higher altitudes are reached.

Hypoxia has progressive effects on the functioning of the central nervous system. Accidents that occur at extreme altitude on Everest and other mountains may be due to poor judgment as a consequence of hypoxic depression of cerebral function. More worrying is that these effects on

cerebral function may be permanent. The American Medical Research Expedition to Everest studied its climbers a year after return to sea level and found some enduring abnormalities of cognitive function and ability to perform fast repetitive movements, although most functions tested had returned to pre-expedition values (Shephard, Bouhlel 1988).

Initially on travelling to high altitude hemoglobin concentrations rise through a fall in the plasma volume due to dehydration. Later, hypoxia stimulates production of erythropoietin by the juxtaglomerular apparatus of the kidney so hemoglobin production increases and hemoglobin concentrations may rise to 200 g/l. The increased viscosity of the blood coupled with increased coagulability increases the risk of stroke and venous thromboembolism. Some authors advocate regular venesection in high altitude climbs; others recommend prophylactic aspirin. Neither has been shown scientifically to reduce the incidence of venous or arterial thrombosis (Peacock 1998,Cremona , Asnaghi , Baderna .2002).

Acute mountain sickness is self limiting and usually affects previously healthy individuals who go too rapidly to high altitude. There may be no symptoms for the first 12-24 hours. Thereafter symptoms develop and usually peak on the second or third day. Symptoms include headache, anorexia, insomnia, and breathlessness. The cause of acute mountain sickness is not understood but is clearly related to hypoxia and factors such as effort, air temperature, previous viral respiratory tract infection and innate susceptibility. Work at Pheriche, Nepal (4343 m) in 1979 found that 43% of trekkers passing through were experiencing headache, anorexia, insomnia, and breathlessness (Peacock 1998, Beall 2001).

High altitude pulmonary edema is a life threatening condition may or may not be preceded by symptoms of acute mountain sickness. Breathlessness increases progressively accompanied by a cough productive of white sputum, which is occasionally tinged with blood. Examination will reveal cyanosis and a mild fever (no more than 38.5°C). Left untreated this condition can progress rapidly and be fatal. The cause is unknown, but it is related to hypoxic pulmonary vasoconstriction. Those affected have a high pulmonary artery pressure and excess hypoxic pulmonary vasoconstriction at sea level. Calcium antagonists and vasodilators can both treat and prevent high altitude pulmonary edema (Peacock 1998, di Prampero & Ferretti 1990).

High altitude cerebral edema is the more malignant form of acute mountain sickness. The symptoms can mimic those of hypothermia, and body temperature should be measured if there is any doubt. If left untreated patients will become unconscious and die (Shephard and Bouhlel 1988, Wagner 2000).

CHAPTER III

MATERIALS AND METHODS

3.1. Study Design

The design for the study was **Within Subjects (repeated measures) Experimental Design**. The Independent variable for the study is mountain trekking and the dependent variables are Heart rate, Blood Pressure, O2 saturation and Acute Mountain Sickness.

3.2. Materials Required

The following materials were used for the research.

3.2.1. Sphygmomanometer

3.2.2. Watch

3.2.3. Pulse Oximeter

3.2.4. Acute Mountain Sickness (AMS) Scale (Annex-I)

3.3. Inclusion criteria's

The following were the inclusion criteria's

3.3.1. Mountain trekkers

3.3.2. Age Group 20- 40 years

3.4. Exclusion Criteria's

The following were the exclusion criteria's

3.4.1. History of Diabetes

3.4.2. Angina

3.4.3. Myocardial Infarction

3.4.4. Infections

3.4.5 Fever

3.5. Procedure:

The study was conducted for 400 trekkers between November, 2006 till November,2010. The average age group was 21.78 ± 1.08 year. 244 males and 156 females participated in the research study. The research study was conducted at a popular trekking spot Doddaribetta, Charmadi Ghats (4,300 ft above sea level).

In the study the physiological parameters were measured prior to trekking and while trekking. The trekkers were explained about the research and written consent was taken from them. The measured parameters were Systolic Blood pressure, Diastolic Blood Pressure, Pulse rate and Oxygen saturation. The sphygmomanometer was used to measure the blood pressure. Pulse Oximeter was be used to measure the Oxygen saturation. The Heart rate was measured by palpation of the radial artery of left hand.

Immediately after the trekking was over a questionnaire named Lake Louise Acute Mountain Sickness (AMS) Questionnaire was given to check for acute mountain sickness. The questionnaire measures for Headache, Gastro Intestinal Problems, Fatigue/weak, Dizzy/lightheaded, Difficulty sleeping, Change in mental status, Ataxia (heel to toe walking) and Peripheral edema.

Fig.3.1. The Blood Pressure and the O2 saturation is measured

Fig.3.2. The data is collected for the Acute Mountain Sickness.

Fig.3.3. Panoramic View of trekking spot Doddaribetta

CHAPTER IV

RESULTS AND DISCUSSIONS

The following are the descriptions of the data collected. We found a significant increase in the Systolic blood pressure, diastolic blood pressure, pulse rate and acute mountain sickness. The oxygen saturation was found to be decreased.

Table.4.1. Details of Pre and Post Measured parameters

Measured Parameters		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Systolic Blood Pressure (Pre)	117.2650	400	16.63766	.83188
	Systolic Blood Pressure (Post)	140.1000	400	17.90157	.89508
Pair 2	Diastolic Blood Pressure (Pre)	79.3975	400	12.40359	.62018
	Diastolic Blood Pressure (Post)	91.9075	400	14.44075	.72204
Pair 3	Pulse Rate(Pre)	88.6750	400	14.19288	.70964
	Pulse Rate(Post)	123.7100	400	16.71240	.83562
Pair 4	Oxygen saturation (Pre)	98.0375	400	1.50224	.07511
	Oxygen saturation (Post)	96.8375	400	1.73454	.08673
Pair 5	Acute Mountain Sickness (Pre)	.0000	400	.00000	.00000
	Acute Mountain Sickness (Post)	1.6500	400	1.66265	.08313

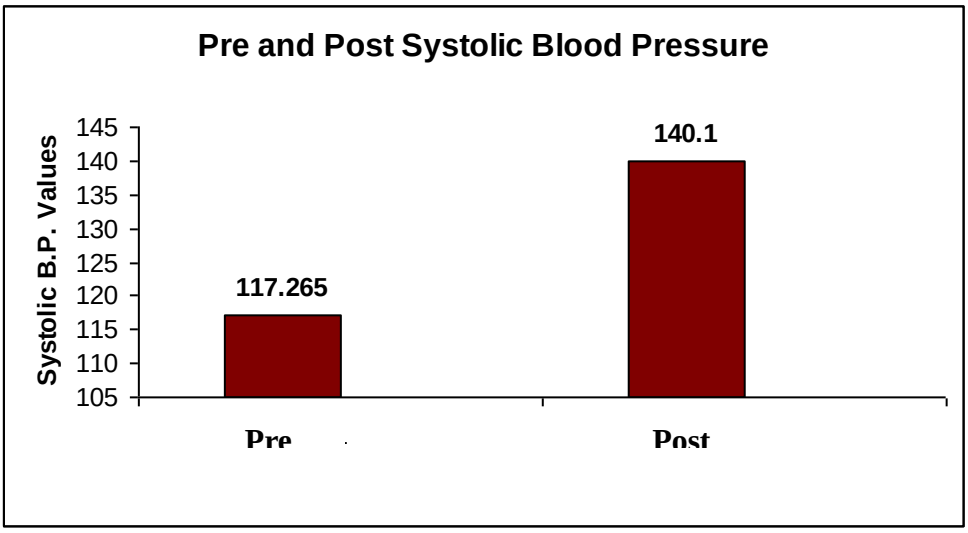

Fig 4.1. Showing the variations of Systolic Blood Pressure before and after trekking.

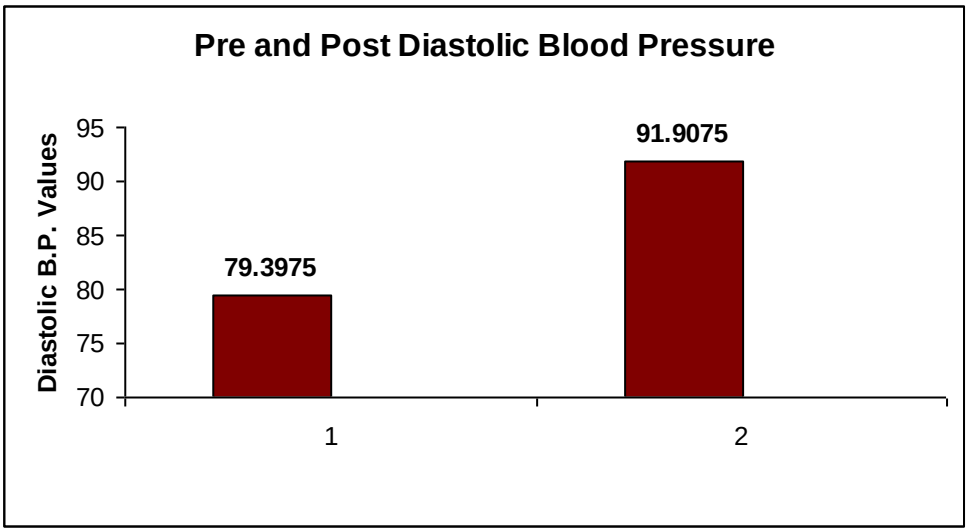

Fig 4.2. Showing the variations of Diastolic Blood Pressure before and after trekking.

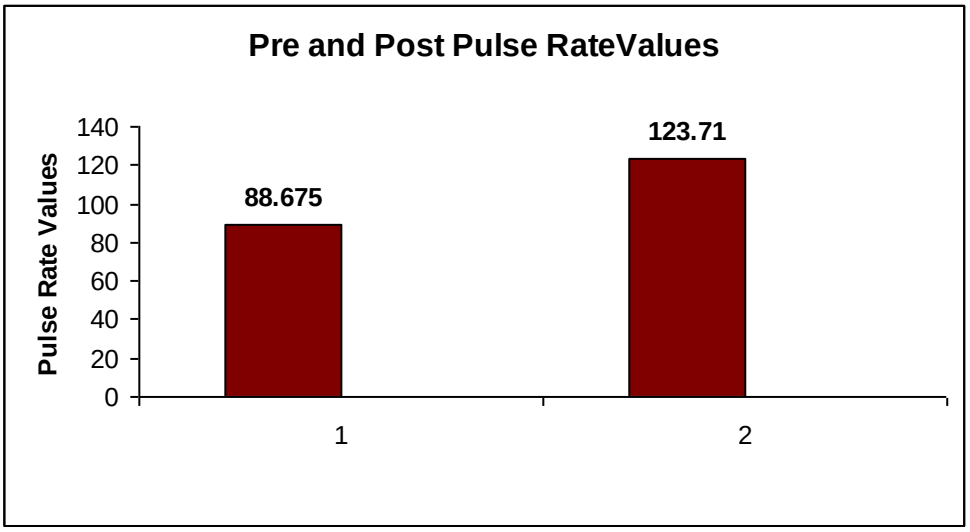

Fig 4.3. Showing the variations of Pulse rate before and after trekking.

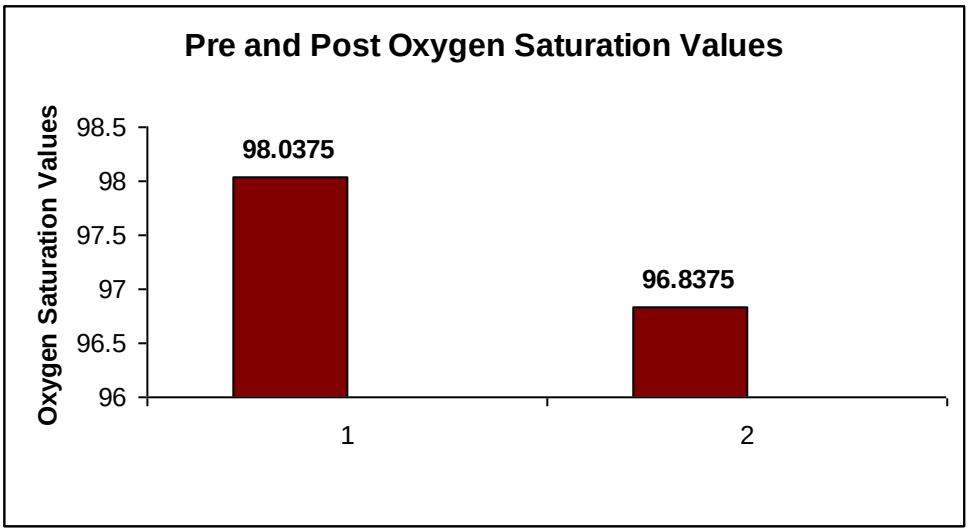

Fig 4.4. Showing the variations of Oxygen Saturation before and after trekking.

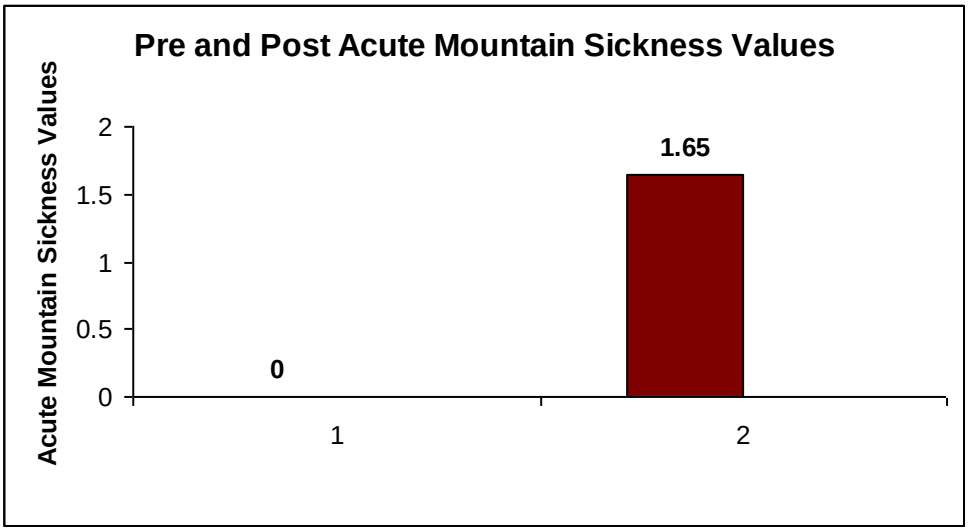

Fig 4.5. Showing the variations of Acute Mountain Sickness before and after trekking.

		N	Correlation	Sig.
Pair 1	Systolic Blood Pressure (Pre and Post)	400	0.273	0.000
Pair 2	Diastolic Blood Pressure (Pre and Post)	400	0.161	0.001
Pair 3	Pulse Rate(Pre and Post)	400	0.272	0.000
Pair 4	Oxygen Saturation (Pre and Post)	400	-0.008	0.870
Pair 5	Acute Mountain Sickness(Pre and Post)	400	0.000	0.000

Table 4.2. Pre and Post Paired Samples Correlations

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	**Systolic Blood Pressure (Pre and Post)**	-22.8350	20.84332	1.04217	-24.8838	-20.7862	21.911	399	.000
Pair 2	**Diastolic Blood Pressure (Pre and Post)**	-12.5100	17.45958	.87298	-14.2262	-10.7938	14.330	399	.000
Pair 3	**Pulse Rate(Pre and Post)**	-35.0350	18.75655	.93783	-36.8787	-33.1913	37.358	399	.000
Pair 4	**Oxygen Saturation (Pre and Post)**	1.2000	2.30397	.11520	.9735	1.4265	10.417	399	.000
Pair 5	**Acute Mountain Sickness(Pre and Post)**	-1.6500	1.66265	.08313	-1.8134	-1.4866	19.848	399	.000

Table 4.3. Showing the Paired Samples Test comparing before and after trekking parameters.

In the AMS score 74 trekkers had no complains, 167 complained of ataxia and 160 complained of Headache.

Data was analysed by **Paired T-Test.**

Systolic BP
Mean: 22.8350
t-value: 21.911
P=0.000

Diastolic BP
Mean: 12.5100
t-value: 14.330
P=0.000

Pulse Rate
Mean: 35.0350
t-value: 37.358
P=0.000

O2 Saturation
Mean: 1.2000
t-value: 10.417
P=0.000

Acute Mountain Sickness
Mean: -1.6500
t-value: 19.848
P=0.000

DISCUSSIONS

4.1. General Introduction

In the study we found a remarkable increase in the blood pressure and pulse rate. The increase in the B.P and pulse rate is due to the increase in the metabolic needs of the muscles (Subudhi and Lorenz, 2008). The metabolic needs increases the cardiac output and that increases the blood pressure. The metabolic needs also increases the pulse rate. The following three mechanisms controls the blood pressure.

4.2. Baroreceptor reflex: Baroreceptors detect changes in arterial pressure and send signals ultimately to the medulla of the brain stem. The medulla, by way of the autonomic nervous system, adjusts the mean arterial pressure by altering both the force and speed of the heart's contractions, as well as the total peripheral resistance. The most important arterial baroreceptors are located in the left and right carotid sinuses and in the aortic arch.

4.3. Renin-angiotensin system (RAS): This system is generally known for its long-term adjustment of arterial pressure. This system allows the kidney to compensate for loss in blood volume or drops in arterial pressure by activating an endogenous vasoconstrictor known as angiotensin II.

4.4. Aldosterone release: This steroid hormone is released from the adrenal cortex in response to angiotensin II or high serum potassium levels. Aldosterone stimulates sodium retention and potassium excretion by the

kidneys. Since sodium is the main ion that determines the amount of fluid in the blood vessels by osmosis, aldosterone will increase fluid retention, and indirectly, arterial pressure.

These different mechanisms are not necessarily independent of each other, as indicated by the link between the RAS and aldosterone release. Currently, the RAS system is targeted pharmacologically by ACE inhibitors and angiotensin II receptor antagonists. The aldosterone system is directly targeted by spironolactone, an aldosterone antagonist. The fluid retention may be targeted by diuretics; the antihypertensive effect of diuretics is due to its effect on blood volume.

In the study the oxygen saturation in blood was found to decrease after mountain climbing. This can be explained by the fact that with increase in altitude the atmospheric pressure decreases and this decreases the partial pressure of inspired oxygen hence the driving pressure for gas exchange in the lungs(Peacock 1998).The stress of the altitude is primarily due to the decrease in (PO_2) partial pressure of oxygen and not barometric pressure(Peacock 1998,Honigman , Theis 1993,Wagner 1996,Wagner 2000).

As Oxygen is 21% dry air, the inspired Oxygen pressure is 0.21×(100-6.3)= 19.6 kPa at sea level. The atmospheric pressure is the sum total of the partial pressures of the constituent gases, Oxygen and Nitrogen and also the partial pressure of water vapour (6.3 Pa at 370 C). The reduction of partial pressure leads to arterial hypoxia (Honigman and Theis 1993).

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4.5. Effect Of Atmospheric Pressure

Fig.4.6 A bottle closed at high altitude.

Fig.4.7 Same bottle while being brought down (squeezed due to high pressure down)

Many trekkers complained of acute mountain sickness the possible cause for that is hypoxia, a low hypoxic ventilatory drive, water retention, and increased vascular permeability (Johnson and Rock 1988).

Trekking resulted in significant increase in Systolic Blood Pressure. In hypothesis 1.3.1 it was stated that there will be significant increase in Systolic Blood Pressure after trekking. The result of the research study show similar results hence hypothesis 1.3.1 is accepted.

Trekking resulted in significant increase in Diastolic Blood Pressure. In hypothesis 1.3.2 it was stated that there will be significant increase in Diastolic Blood Pressure after trekking. The result of the research study show similar results hence hypothesis 1.3.2 is accepted.

Trekking resulted in significant increase in Pulse Rate. In hypothesis 1.3.3 it was stated that there will be significant increase in Pulse Rate after mountain climbing. The results of the research study show similar results hence hypothesis 1.3.3 is accepted.

Trekking resulted in significant decrease in Oxygen Saturation. In hypothesis 1.3.4 it was stated that there will be significant decrease in Oxygen Saturation after trekking. The result of the research study show similar results hence hypothesis 1.3.4 is accepted.

Trekking resulted in significant increase in Acute Mountain Sickness. In hypothesis 1.3.5 it was stated that there will be significant change in Acute Mountain Sickness after trekking. The result of the research study shows similar results hence hypothesis 1.3.5 is accepted.

CHAPTER V

CONCLUSION

6.1. Conclusion

6.1.1. Systolic Blood Pressure, Diastolic Blood Pressure and Pulse rate increased significantly post trekking. The statistical values were (Systolic BP: t-value 21.911, P=0.000), Diastolic BP (t-value: 14.330, P=0.000), Pulse Rate (t-value: 37.358, P=0.000).

6.1.2. The Oxygen saturation in the blood decreased significantly post trekking. The statistical value was (t-value: 10.417, P=0.000).

6.1.3. The scores in the acute mountain sickness taken post trekking showed significant change in values. The scores were taken using Lake Louise Acute Mountain sickness Questionnaire. The statistical value was (t-value: 19.848, P=0.000).

6.2. Recommendations

6.2.1. Similar research may be conducted involving large number of subjects.

6.2.2. Similar research may be conducted involving different mountain heights.

6.2.3. Similar research may be conducted on different age groups.

6.2.4. Similar study may be conducted on persons suffering from obesity, diabetes and coronary artery disease.

6.2.5. This study will provide understanding of the physiological parameters for the persons undertaking trekking and also will provide insight for the sports activities performed on high altitude as there is decrease in oxygen saturation with height.

6.2.6. This research study will provide better insight for sports coaches, physiotherapists, diabetologists, cardiologist and nutritionists.

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